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ON THE THEORY OF ACCELERATION TOLERANCE

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Acceleration tolerance is a complex concept which is frequently associated with a certain amount of misunderstanding. Acceleration research may involve different aspects of acceleration tolerance depending upon the major interest of that research, however the entire scope of the tolerance envelope must always be considered. Current operational requirements involving rapid onset, sustained high $+G_z$ makes neurologic $+G_z$-tolerance a prime consideration. A detailed understanding of what is known about neurophysiologic tolerance is therefore a key aspect of basic and clinically oriented research. The $+G_z$-time tolerance curve is discussed in detail, specifically describing what is currently known and what remains to be investigated. A breakdown of the major aspects of acceleration tolerance is suggested as an initial step in unifying the concepts necessary to fully evaluate the required operational tolerance of aircrew flying fighter type aircraft.

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INTRODUCTION

Development of advanced $+G_z$ protection equipment and techniques and potential prevention of $+G_z$-induced loss of consciousness (G-LOC) are highly dependent on accurate determination of normal +G-tolerance and the ability to observe measurable tolerance differences. One of the most useful principles for defining certain aspects of $+G_z$ tolerance, initially developed by German aviation medicine (8) and later refined by Stoll (16) in 1956, is known as the $+G_z$-time tolerance curve. This extremely valuable conceptual curve focused the acceleration research community on the physiologic importance of the rate of onset of $+G_z$. Until that time, the most important, $+G_z$ tolerance parameter was the absolute level of $+G_z$ attainable, as evidenced by reviewing the literature which frequently did not even specify onset rates. The early data covering the more rapid onset of $+G_z$ was only approximate, being derived from the aircraft studies of von Diringshofen, Gauer, Matthes, and Ruff (8). The construction of $+G_z$-time tolerance curves has very rightly been based on experimentally derived data. The Stoll curve was based on 40 experimental points, taken from several different studies. Review of the experimental points used in the curve generated by Stoll reveals that the low-level longer duration (gradual onset) $+G_z$ data was predominately blackout (and greyout) data, whereas the higher level short duration (more rapid onset) $+G_z$ data was predominately unconsciousness data. It was recognized that in the region of 6 G/s a shorter time lag existed between the beginning of greyout and the onset of unconsciousness than at the more gradual onset rates. A table was given, specifically describing the progressively shorter time to unconsciousness following greyout as the rate of $+G_z$ onset increased (16).

Calculations from the tables given result in both the onset of greyout and onset of unconsciousness approaching the ordinate asymptotically as the $+G_z$ onset rate increases. Data in the rapid onset region was not as readily accessible because of the technical difficulties in accurately measuring onset rates inflight and early centrifuges were limited in their rapid onset capabilities.

As aircraft development progressed, the rate of onset of $+G_z$ became more physiologically important because the aircraft were able to not only generate rapid onset $+G_z$ but were able to sustain the high $+G_z$-level for significantly longer periods. Aerospace medical research failed to undertake steps to fully investigate the physiologic aspects of the very rapid onset rates prior to the introduction of aircraft into the operational inventory. The US Air Force's need for a centrifuge with onset rates similar to those in operational aircraft was recognized only as operational losses began to be realized. Even now, the onset rates of existing Air Force centrifuges fail to equal the onset rate capability in existing fighter aircraft. The ability to investigate current and potential future fighter aircraft acceleration envelopes is neither available nor planned. The only capability to investigate acceleration characteristics in advance of operational deployment of aircraft was at the Naval Air Development Center, Warminster, Pennsylvania. Their very advanced electric drive centrifuge, able to generate very rapid onset G, became operational in the early 1950's. One can only question whether it was superior technical insight or other reasons that drove the Navy to build such a superior facility. For the present, that centrifuge facility is the only one capable of investigating the current maximum $+G_z$ fighter aircraft envelopes. Development of advanced methods for solving G-LOC and understanding in-flight phenomenon in the very high onset arena require similar sophisticated research facilities. The design of such facilities and the applied research performed in them should be dictated by a knowledge of the anticipated requirements of future aircraft and combat tactics gained from the aerospace engineering and operational communities.

THE $+G_z$-TIME TOLERANCE CURVE

The $+G_z$-time tolerance curve separates two dramatically different physiologic states that exist during $+G_z$ exposure (see Figure 1). On one side of the curve the central nervous system (CNS) functions normally, representing a state of consciousness. On the opposite side, the entire physiologic reserve of the body has been overcome resulting in a state of unconsciousness. The curve itself possesses a variable

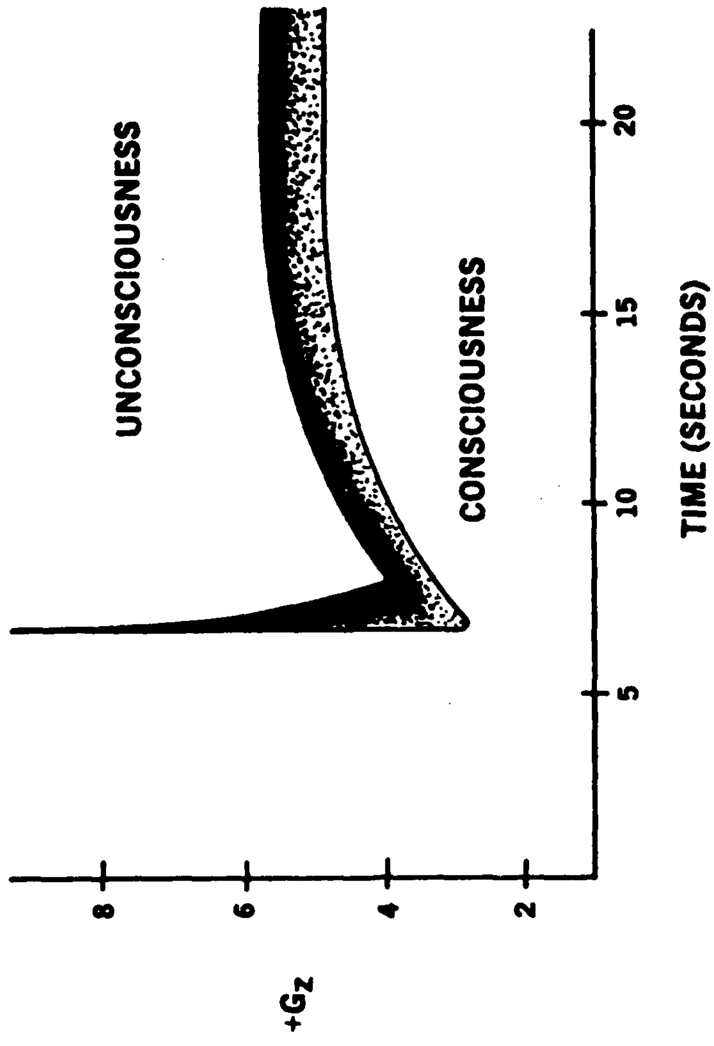

FIGURE 1. The +G_z-Time Tolerance Curve.

width, which represents the visual symptoms which occur during the transition from asymptomatic consciousness to unconsciousness. The edge of the curve on the conscious side begins with progressive loss of vision (greyout), continuing to a point where only central vision remains (tunnel vision). The central vision progressively dims until no vision remains (blackout). Blackout without unconsciousness results from the existence of intraocular pressure (approximately 20 mm Hg) which is higher than the pressure within the brain. As the $+G_z$ level increases, the blood pressure at eye (and brain) level progressively falls. When the blood pressure falls to the point that it cannot enter the eye to perfuse the retina (because the intraocular pressure is higher than the blood pressure) blackout results. The increasing greyout leading to tunnel vision develops because the perfusion pressure progressively drops in going from the central retinal artery outward toward the periphery of the retina. Perfusion in the small peripheral vessels of the retina begins to fail prior to perfusion failure near the central retina artery, causing central vision to be the last area to be maintained. Since the pressure within the brain tissue is not as high as intraocular pressure, perfusion and therefore function, continues in the brain. When the blood pressure finally drops to the point where the brain cannot be adequately perfused, unconsciousness will result. The mechanism of blackout was exquisitely proven by Duane (2, 3) and Newsome (12) through evaluation of the retinal circulation during $+G_z$ stress. The final transition represented by the width of the curve is from blackout to unconsciousness (see Figure 2).

In reality, the $+G_z$-time tolerance curve consists of two separate curves based on physiologic tolerance limits (see Figure 3). One curve is based on cardiovascular tolerance (right side) and the other curve is based on neurologic tolerance (left side). The two curves intersect to form the overall $+G_z$-time tolerance curve that is useful for describing the physiologic response and tolerance to $+G_z$ stress. It should be emphasized that the curve does not cover the longer (20 seconds) fatigue limited tolerance (both physiologic and physical fatigue) to prolonged duration $+G_z$ stress.

DESCRIBING LOSS OF CONSCIOUSNESS

Before describing the various aspects of the $+G_z$-time-tolerance curve, it is necessary to develop an understanding of what is known about G-LOC (19). The exact, physiologic mechanism and anatomic site within the CNS which determines the onset of unconsciousness resulting from inadequate blood flow is unknown. A detailed description of what is known about G-LOC is required before a logical solution to the problem can be achieved.

FUNCTIONAL AND INTEGRITY BUFFER PERIODS

Anoxia/hypoxia leads to a continuous process of disruption of neuronal functioning. It begins with an alteration sufficient to prevent the normal biochemical processes necessary for the integrated functioning of the cells within the CNS. This may begin in several regions of the brain almost simultaneously because $+G_z$ acceleration probably produces a global redirection in blood flow away from the brain. If this redirection in blood flow becomes sufficiently severe, it results in the loss of integrated function of the brain. When the rate-limiting G-LOC trigger site becomes dysfunctional, the body's protective mechanism is for unconsciousness to occur. Teleologically, this is the appropriate evolutionary mechanism for a biped to ensure protection of the brain. Unconsciousness results in the body becoming totally relaxed in the earth's gravitational field. The resulting body position assumed is horizontal, (lying supine or prone), which places the brain at the same level in the gravitational field as the rest of the body (heart). This position maximally facilitates perfusion of the brain.

Even though the individual is unconscious, much of the brain, the areas most resistant to the lack of blood flow and those which have not lost a critical amount of blood flow, may continue to function normally. The cells all have, albeit small in some cases, a buffer period (functional buffer period) to ensure some tolerance to lack of blood flow. Once this period is exceeded, normal function ceases. For opera-

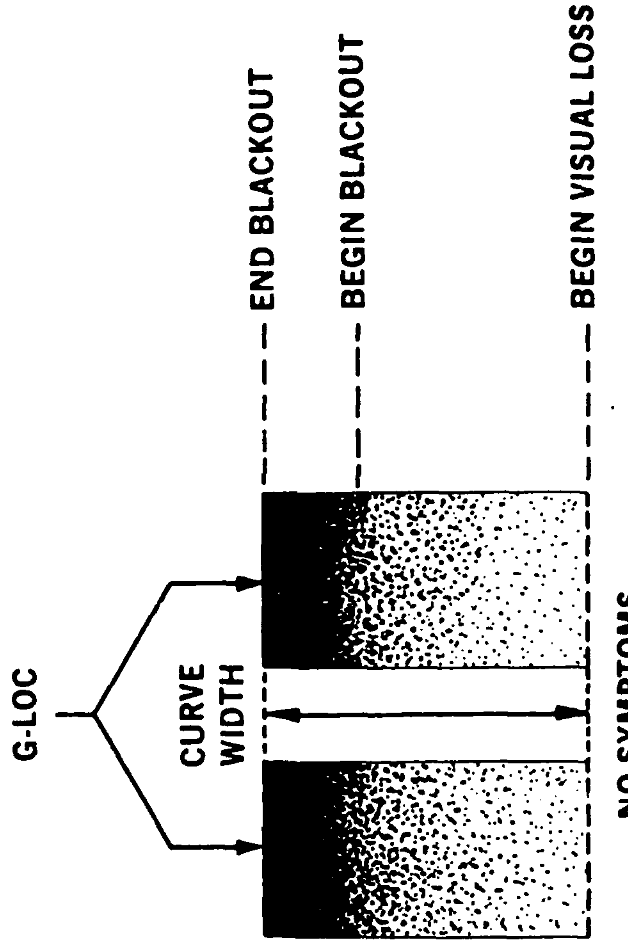

FIGURE 2. The Width of the $+G_z$-Time Tolerance Curve in the Cardiovascular Region.

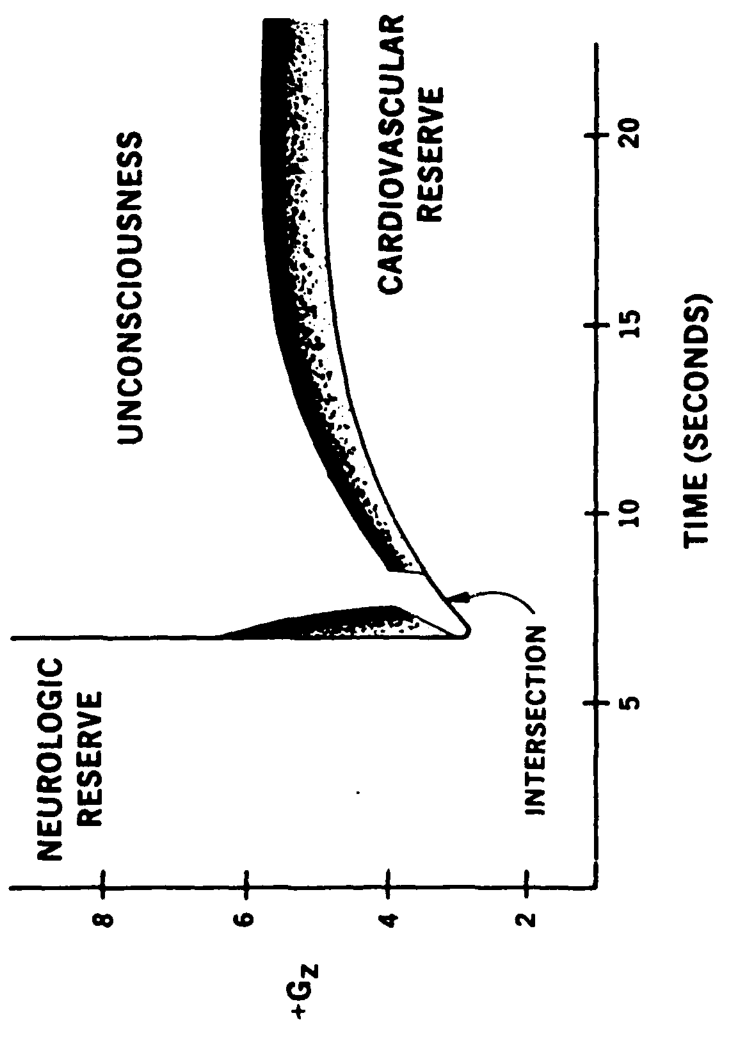

FIGURE 3. The +G_Z-Time Tolerance Curve Consists of Two Separate Curves Based on Cardiovascular Reserve and Neurologic Reserve.

tional purposes this period has been referred to as the CNS reserve or oxygen reserve. If blood flow is restored, then normal cell function will return, provided the next buffer period (integrity buffer period) is not exceeded. If the integrity buffer period of a cell is exceeded, then the cell will suffer damage. CNS cells are extremely delicate and any cellular damage is usually considered irreparable.

We shall therefore define the buffer periods described above in the following manner. The functional buffer period is the length of time the brain continues to function without adequate cerebral blood flow. The time of the functional buffer period for rapid onset $+G_z$ acceleration (anoxic/hypoxic anoxia) is approximately five to seven seconds in man. The integrity buffer period is the length of time the brain (or any portion of the brain) can tolerate inadequate blood flow without structural damage. Both periods actually begin at the moment cerebral blood flow becomes inadequate, however we will consider the integrity buffer period begins at the moment overall normal function fails. Summation of the functional buffer period and the integrity buffer period, as defined, is the absolute amount of time the brain can tolerate inadequate cerebral blood flow without structural damage (Figures 4 and 5). The exact time of the integrity buffer period is uncertain, but it is on the order of six minutes in dogs. A multitude of clinical experience suggests 2 to 3 minutes is the allowable time in humans for assured complete recovery. The duration of the functional and integrity buffer periods is dependent on the exact type of anoxic/hypoxic challenge. Experimentation to define these buffer periods in humans is either difficult (functional buffer period) or immoral (integrity buffer period), since LOC and permanent damage or death are only separated by an unknown (and perhaps infinitesimally small) amount of time.

The recovery process is equally important because enhanced pilot recovery is critical to aircraft recovery. The recovery process may well be dependent on the length of the excursion into the integrity buffer period. The deeper (longer) the excursion into the integrity buffer period lasts, the longer the time it will take to regain normal function. Gross physiologic functional recovery is relatively rapid (seconds), however complete psychophysiologic recovery maybe extended (minutes to hours). The exact relationship however is unknown (Figure 6).

THE CARDIOVASCULAR $+G_z$ TOLERANCE CURVE

If given adequate time, the cardiovascular system is able to mount a defensive response to the acceleration stress resulting in overall $+G_z$ tolerance being enhanced (see Figure 7). $+G_z$ tolerance therefore increases in relation to the adequacy of cardiovascular reflexes. The importance of cardiovascular responses begins when the $+G_z$ exposure level is sufficiently high such that resting blood pressure is not high enough to provide adequate perfusion of the brain. Lambert and Wood very nicely defined the relationship between eye-level blood pressure and the physiologic symptoms which result from $+G_z$ stress (10). They found the decrease in arterial blood pressure to be approximately 30 mm Hg per G (32 mm Hg/G exactly). (The currently used value is 22 mm Hg per G.) In addition, they and others correlated the physiologic symptoms with eye-level blood pressure as shown in Table 1 (10,15). From these studies it may be assumed that the onset of symptoms (initiation of greyout) would result when the $+G_z$ level is high enough to reduce eye-level blood pressure below 50 mm Hg. Assuming a systolic blood pressure of 120 mm Hg, an increase in 2.2 G_z (to a $+G_z$ level of 3.2) should be sufficient to enter the earliest phase of peripheral light loss based on resting conditions (i.e., no cardiovascular reflexes). Blackout would similarly begin with an increase in 3.1 G_z (to a $+G_z$ level of 4.1) and ends with G-LOC occurring with an increase in 3.8 G_z (to a $+G_z$ level of 4.8) (see Figure 8). Therefore, in the average man, without cardiovascular reflexes, this is what hydrostatic principles would predict if we solely assume that the spectrum of limiting symptoms of the eye and brain are the result of inadequate blood flow.

Since we do have cardiovascular reflexes which result in an overall adaptive response to counteract the excess $+G_z$ forces, we would expect some type of increase in the level at which these limiting symptoms occur. The next question is related to time. How rapid do the cardiovascular reflexes respond

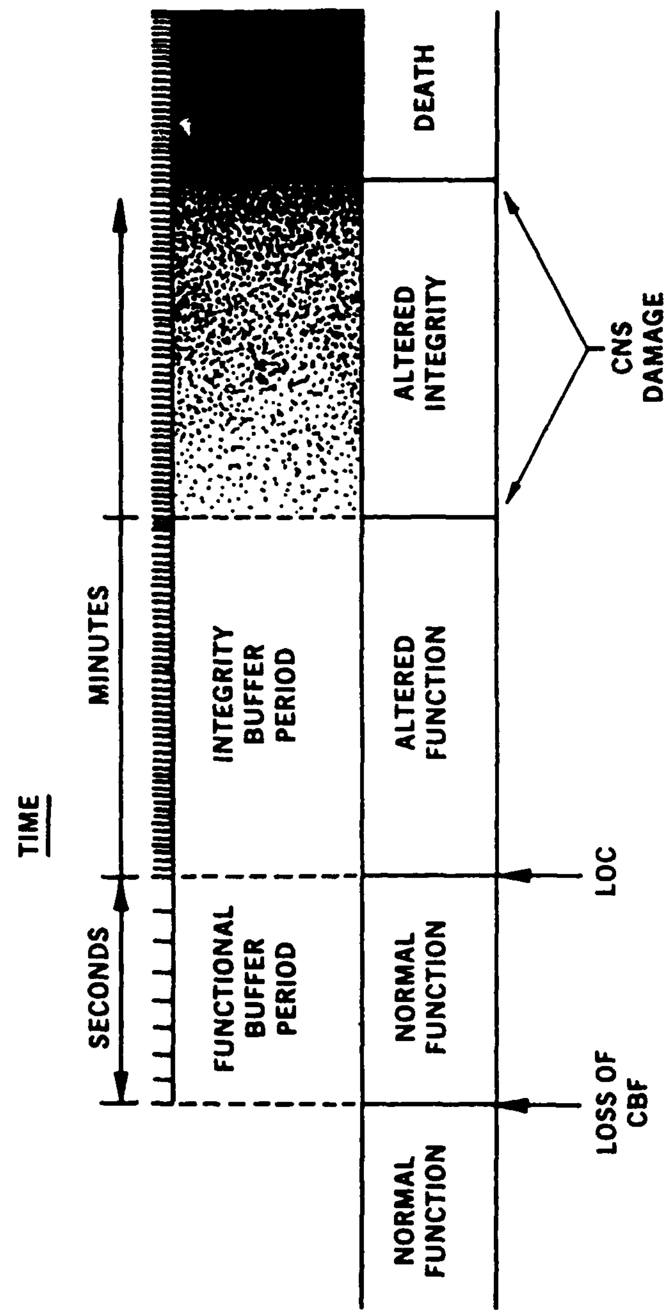

FIGURE 4. The *Continuum* of Pathophysiologic Events Resulting from Inadequate Cerebral Perfusion.

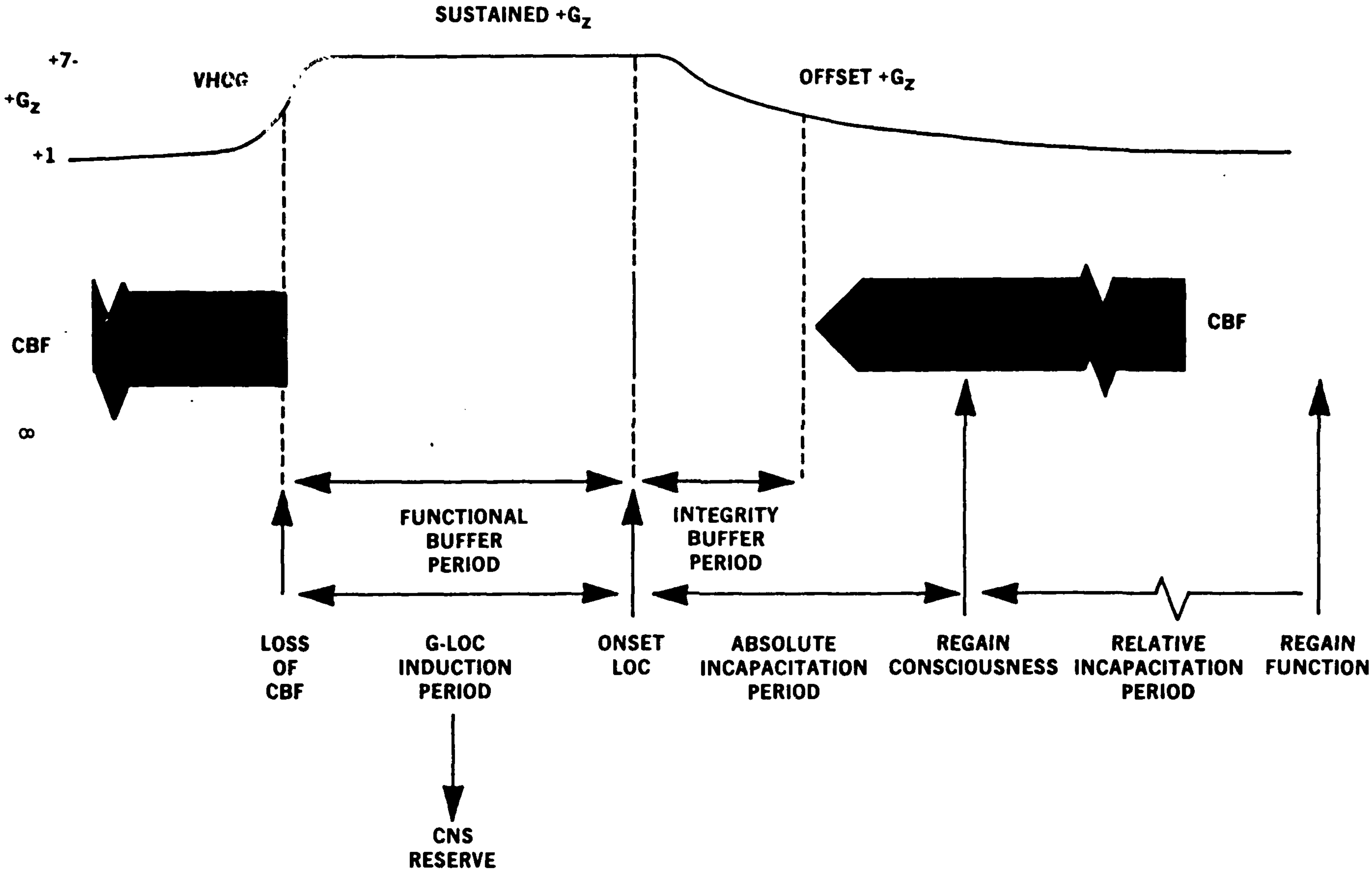

FIGURE 5. The Relationship of $+G_z$-Induced Loss of Consciousness and Recovery with the Pathophysiologic Continuum.

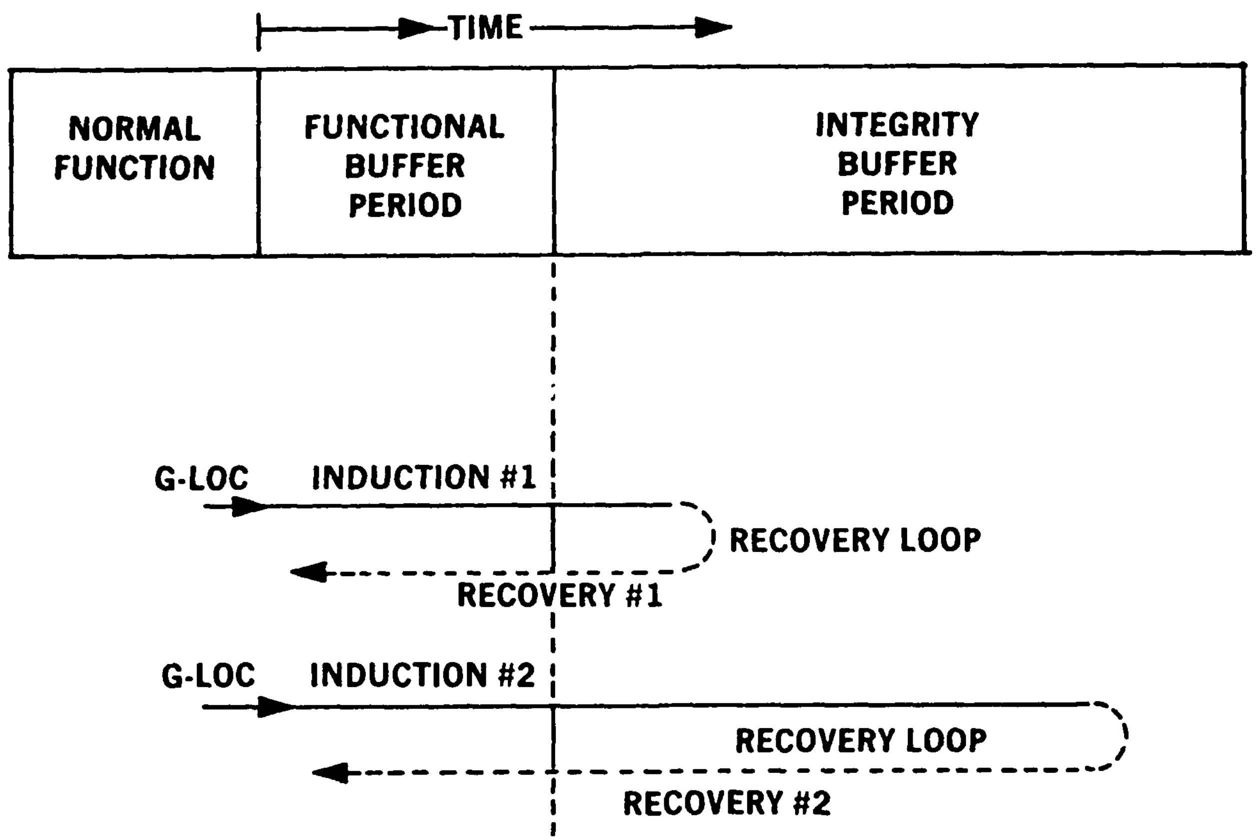

FIGURE 6. Theoretical Relationship Between Depth of Penetration into the Integrity Buffer Period and G-LOC Recovery.

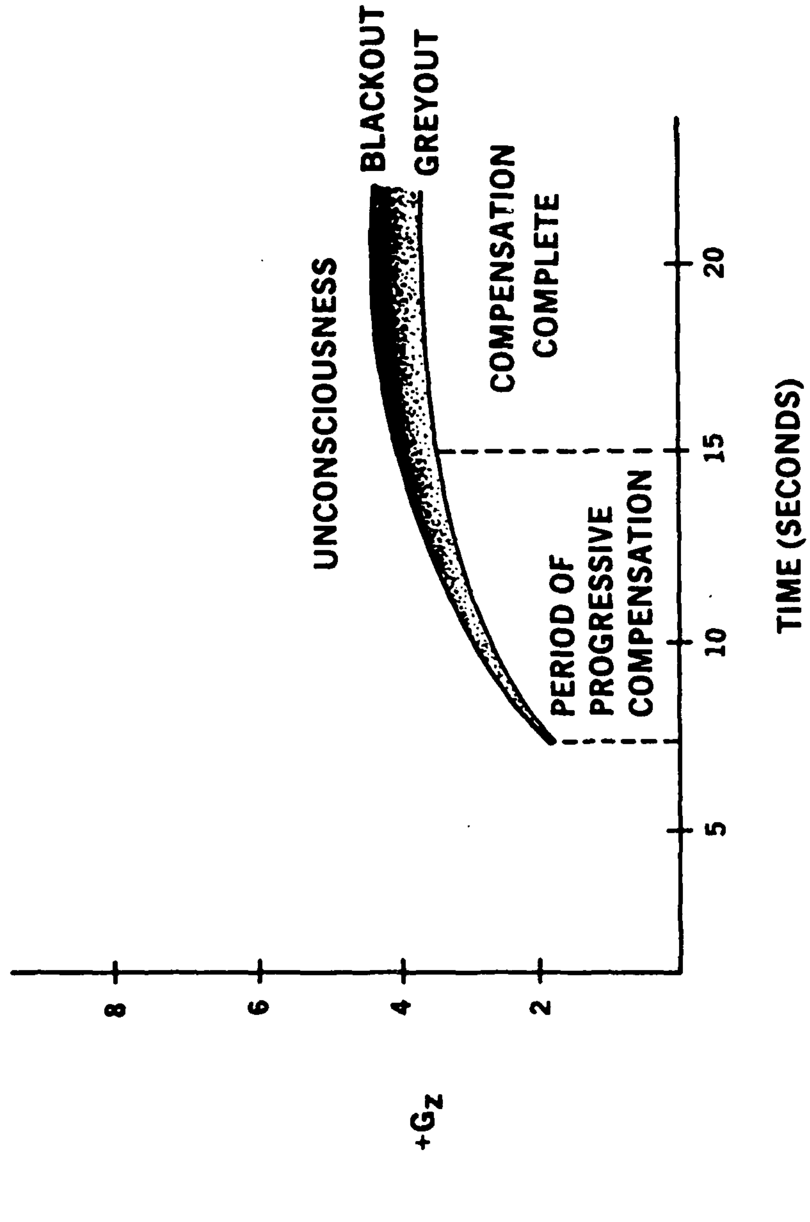

FIGURE 7. The Cardiovascular +G_z-Tolerance Curve.

Table 1. The relationship between eye-level blood pressure and visual symptoms.

	VISUAL/NEUROLOGIC SYMPTOMS	EYE-LEVEL SYSTOLIC BP (mm Hg)
I. Duane*	Visual dimming	30-49
	Peripheral light loss	20-32
	Blackout	0-21
	G-LOC	<0
II. Lambert+	No symptoms	>50
	Peripheral light loss	<20
	G-LOC	0

* Reference 3
\+ Reference 10

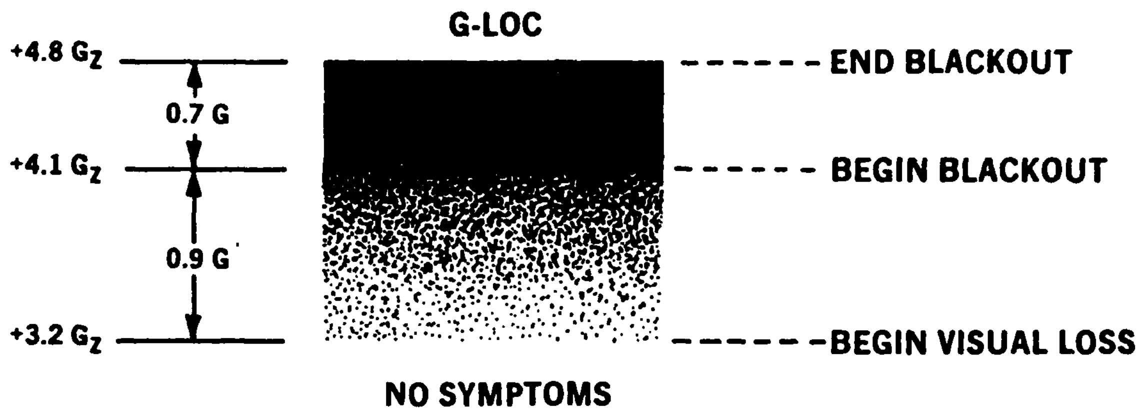

FIGURE 8. Relaxed Cardiovascular $+G_z$-Tolerance Curve Composition Based on Symptoms.

to $+G_z$ stress? We must coincidently ask, what is the rate of onset of the $+G_z$ stress that the cardiovascular reflexes can and cannot respond to? Using the change in heart rate as a convenient measure of cardiovascular reflex response, we can determine the maximal cardiovascular response rate. Blood pressure response is more appropriate but more difficult to obtain universally. First, if the $+G_z$ onset is slow enough, the heart rate will continuously change coincidently with the change in $+G_z$. Experimental work has not exhaustively investigated this question, however sufficient data is available to make estimations. It is well known that $+G_z$ onset rates in the range of 0.07-0.1G/s are sufficiently slow to allow continual compensation of the heart rate in response to the $+G_z$. The exact $+G_z$ onset rate at which the heart rate begins to lag behind the $+G_z$ onset has not been thoroughly investigated. We can, however, take the approach of defining how long it takes the cardiovascular reflexes to respond to very rapid $+G_z$ onset rates to a high enough $+G_z$ level to maximally induce cardiovascular reflexes. This has been evaluated by comparing the heart rate change during slow onset $+G_z$ (0.1G/s) (slow enough for a continual change in heart rate as the $+G_z$ increases up to maximal heart rate response) with very rapid onset $+G_z$ stress (6G/s) to the same $+G_z$ level with the heart rate response again reaching the same maximum. These experiments have revealed that (up to a 6G/s onset rate) it takes approximately 30 s. for cardiovascular reflexes to maximally respond (6,7). The majority of the compensatory response is complete by 15s. This agrees with the time course of instantaneous heart rate changes in normal subjects in response to standing ($+1G_z$ stress) where the mean heart rate peak occurred at 12s (18). These investigators used 15s for practical purposes as the time for peak heart rate to be achieved as a result of standing. Other researchers have relied on 10s as being long enough for reflex activation (4). It therefore appears reasonable to define a gradual $+G_z$ onset rate (GOR) to be a rate which has as an upper limit, a maximal $+G_z$ level that can be reached without symptoms within l5s, i.e., 3G/15s (0.2G/s). A large portion of the USAF School of Aerospace Medicine (USAFSAM) centrifuge GOR data was generated utilizing 1G/15s (0.07 G/s) but has been recently altered to a GOR of 1G/10s (0.1G/s). Both of these onset rates, well below O.2G/s, have been utilized to evaluate the normalcy of cardiovascular responses in experimental studies and aeromedical evaluation of aircrew. After 15s the cardiovascular reflex response is essentially maximal and no further $+G_z$-tolerance augmentation occurs. The $+G_z$-time tolerance curve (cardiovascular region) therefore reaches a maximum around 15s and remains flattened thereafter.

Research comparing the reflex heart rate response to different $+G_z$ onset rates reveals that in transitioning from gradual onset (0.1G/s) to rapid onset (1.0G/s) and then to very high onset (6.0G/s) the cardiovascular reflex response time is totally inadequate (too slow) for onset rates in the 1.0 to 6.0 G/s onset range (6,7). The change in heart rate upon reaching the maximum $+G_z$ level during 1.0G/s acceleration exposures is only one-half of the maximum response required for that specific level of $+G_z$, and for 6.0G/s exposures only a change of one-fourth that required. The operational $+G_z$ onset rates in current aircraft (as rapid as 9 G/s in the F-16) therefore greatly exceed the ability of the cardiovascular reflexes to initially support the pilot. The more rapid the onset rate, the less cardiovascular reserve available to enhance tolerance (until mobilized at 15s). The left-hand side or neurologically determined portion of the curve is therefore the operationally important area when considering rapid onset $+G_z$. As previously mentioned, much of the $+G_z$-time tolerance curve does have a finite width, reaching a maximum width for the gradual onset acceleration rates. The curve width, as shown in Figures 2 and 8, is defined on the low side by the onset of visual symptoms initiating greyout and on the high side by the end of blackout resulting in G-LOC. Stauffer's laborious studies give an indication of curve width even though the onset rates were not exactly specified (15). The mean difference from the onset of greyout to G-LOC was 1.4 G-units (O.6G being progressive greyout followed by 0.8G of blackout preceding G-LOC). Lambert's data is very similar as shown in Figure 8. In the region of the curve defined by gradual onset acceleration the time-width is determined by the rate at which the decline in the 50 mm Hg of eye-level blood pressure occurs (50 mm Hg to 0 mm Hg) and the CNS tissue functional buffer period (15).

As the acceleration onset rates increase, eye-level blood pressure is overcome progressively more rapidly as the cardiovascular reflexes are unable to keep up. Eventually the onset rate is so rapid that cardiovascular reflexes are initially completely overcome but recover before symptoms occur. Finally, the acceleration onset rate is so rapid that the eye-level blood pressure is immediately and continuously reduced (sustained $+G_z$) to 0 mm Hg. The acceleration onset rates that overcome the cardiovascular reflexes and eventually lead to the level where $+G_z$- tolerance is strictly determined by the CNS functional buffer period are rapid onset rates (ROR). In the ROR range, $+G_z$-tolerance transitions from a dependence on the cardiovascular system to a dependence on the CNS functional buffer period (neurologic system). Integrating the two separate curves for cardiovascular and neurologic $+G_z$-tolerance results in an overlapping $+G_z$-time tolerance curve (which is operationally useful) with a dip in the area of intersection, as shown in Figure 3.

THE NEUROLOGIC $+G_z$ TOLERANCE CURVE

The neurologic tolerance to $+G_z$, represented by the left side of the $+G_z$-time tolerance curve, results from reaching a sustained $+G_z$ level above tolerance so quickly that there is not time for adequate cardiovascular reflex response (see Figure 9). Symptoms therefore become dependent on the length of time that the retinal and brain tissues can function without adequate perfusion. Once these tissues fail to receive adequate perfusion how long they continue to function before failing is the neurologic reserve or the functional buffer period as previously discussed. Some basic physiologic questions arise at this point. It is evident that onset rates on the order of 3G/s to 6G/s or more reduce the blood flow to an inadequate (or no flow) level within 1-2s. Symptoms then become apparent based on how long the retina will continue to function without blood flow and likewise the same for the brain tissues. Do the retina and the brain have the same reserve, (i.e., do they fail at the same time if both fail to receive adequate perfusion simultaneously)? The fact that intraocular pressure is higher than intracranial pressure would not appear to be an important differential factor when the perfusion is almost instantly overcome in both the retina and the brain. It has been shown that the retina is the body's most dependent tissue for a continuous supply of oxygen to maintain function, having a measured metabolic rate four times greater than even the grey matter of the brain (17). Livingston pondered this question, pointing out the necessity to define the effects of the abrupt cessation of blood flow to the various areas of the central nervous system (11). Duane confirmed the extreme sensitivity of the retina to acceleration induced anoxia, indicating that it would not function more than 2-5s after blood supply was impaired (2,3). In addition, he indicated that retinal function was regained 2-5s after its source of oxygen was restored. It is unclear whether he considered the retina and brain to be equally sensitive to the abrupt cessation of blood flow. It has been pointed out that specific cells, the ganglion cells and nerve fibers of the inner retinal layers, were extremely sensitive to anoxia (13). G-LOC was stated to occur in less than 4s. Others have stated that the time reserve (now defined as the functional buffer period) of the nervous tissue was 2s (8). Duvoisin utilized the Valsalva maneuver to induce loss of consciousness and found that 6s of hypotension resulted in unconsciousness (5). Newsom's investigations into the retinal circulation suggested that greyout began when the eye-level diastolic blood pressure fell below the intraocular pressure with blackout occurring sequentially after eye-level systolic blood pressure fell below the intraocular pressure (12). Overall this would indicate that the retina was even sensitive (and detectability so) to the very short intermittent lapses in perfusion between heart beats (during diastole). This diastolic time period is very short at the high heart rates induced by $+G_z$ stress.

Almost instantaneous arrest of the cerebral circulation was achieved by Kabat using a cervial neck occlusion cuff (14). The results using this technique revealed that eye fixation occurred in almost half of the normal subjects in 5.5s (range 4.0-10.0s), with LOC occurring approximately 1.0s later. The average time from arrest of cerebral circulation to loss of consciousness in normal young men was 6.8s. Most subjects reportedly had visual symptoms prior to LOC.

It therefore seems probable that the retina has a greater sensitivity to acute reduction in blood flow than the brain and the sensitivity differential between the retina and brain is great enough to be

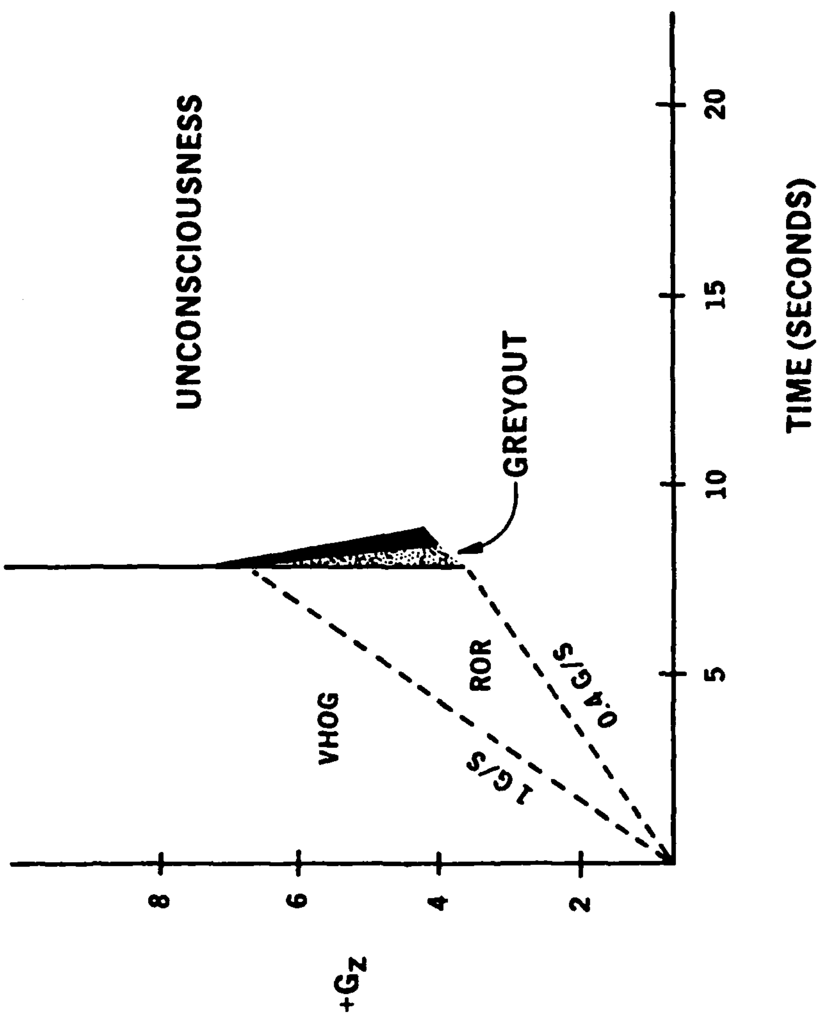

FIGURE 9. The Neurologic $+G_Z$-Tolerance Curve.

physiologically measured. Although physiologically significant, the short (approximately 1s) time differential is unlikely to be of operational significance for fighter aircrew. That is, visual symptoms which could be used as a warning for impending loss of consciousness during abrupt loss of cerebral blood flow would not be of use to aircrew. The lack of visual warning has been unquestionably proven during centrifuge experiments and in-flight in a multitude of reports. A potential sequence which arises in these G-LOC observations is related to the subject suffering G-LOC being able to remember the visual loss prior to G-LOC upon recovery. The retina may actually fail first, however, it may not be registered into memory by the brain and therefore is forever lost to memory as G-LOC occurs. As such it would not be remembered. If this is indeed what actually occurs, then the neurologic $+G_z$-tolerance curve width may be divided into 2 bands, Figure 10. During less rapid onset rates, it consists of both a band for recognizable symptoms and a band for unrecognizable symptoms. As the onset rates become more rapid, the recognizable symptom band narrows until at very high onset rates the curve width consists of only an unrecognizable symptom band which separates consciousness and unconsciousness. The band width being due to a differential sensitivity to acceleration induced anoxia, if it indeed exists.

From a physiological standpoint it is evident that both the retina and brain have a finite reserve which was previously defined as the functional buffer period. When the $+G_z$ onset rate is so rapid that it immediately causes lack of perfusion to the retina and brain, a finite limiting time (the functional buffer period) defines the $+G_z$-time tolerance curve. Based on the cerebral blood flow theory alone, the curve would become fixed on the time axis based on the neurologic tissue functional buffer period. At some onset rate the width of the curve would also become fixed, defined by the length of time of visual symptoms exist prior to onset of G-LOC. As confirmed by rapid onset $+G_z$ stress during centrifuge and in-flight G-LOC without preceding visual symptoms, for operational purposes, the width of the symptom curve disappears. At high enough onset rates the transition from no symptoms to G-LOC is instantaneous and is dependent only on the functional buffer period (the curve has no observable width). Considering the cervical neck occlusion studies (14), explosive decompression studies, and the centrifuge experiments using very high onset $+G_z$, the $+G_z$-time tolerance curve evidently becomes fixed at approximately 7s. It is vertical at that time and does not approach the ordinate asymptotically as previously described by Stoll (16). The point on the curve where no recognizable curve width exists may be considered the dividing point between rapid onset (ROR) and very high onset $+G_z$ (VHOG), see Figure 10.

This neurologic reserve requirement for definition of the curve assumes that no other mechanisms are present. For instance, if very high onset rates of $+G_z$ result in increased CNS metabolism, the curve may shift more toward the ordinate as the neurologic reserve is depleted in an increasingly more rapid rate. Some acceleration experts consider that 7s of neurologic reserve is (slightly) too long, with 5s being a more accurate value (9). The 7s time is, however, consistent with currently available centrifuge data and agrees with the cervical neck occlusion studies and explosive decompression altitude studies. These exposures are not exactly the same as acceleration, however they do agree quite closely. More precise values should be confirmed with experimentation involving variable onset $+G_z$ rates. These experiments with VHOG exposures will almost assuredly involve "no warning" G-LOC as a frequent and inevitable endpoint. An exact definition of the functional buffer period is very important both operationally and for basic research aimed at prolonging this period. $+G_z$ neurologic tolerance is therefore defined by the functional buffer period.

Extending the functional buffer period would have more than a single $+G_z$ tolerance enhancing benefit as can be understood by examining the overall $+G_z$ time tolerance curve (Figure 11). First, it would extend the length of time the CNS could function without adequate blood flow, thereby preserving consciousness by increasing the functional buffer period. Second, any extension would benefit the time available for the cardiovascular reflexes to respond. If a suitable CNS capacitor could extend the functional buffer period, by as little as seven seconds, the cardiovascular reflexes would be essentially fully

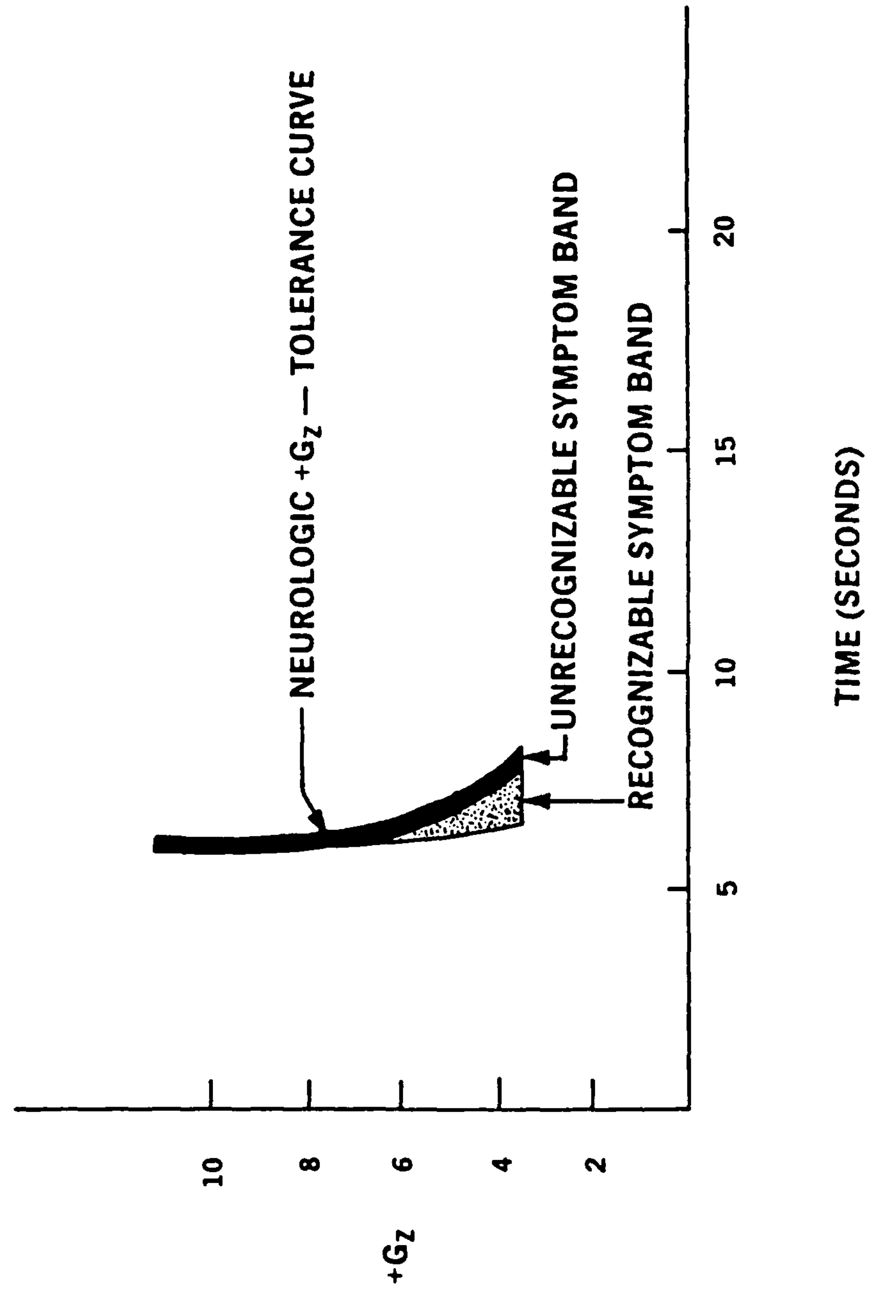

FIGURE 10. Theoretical Composition of the Neurologic +G_Z-Tolerance Curve.

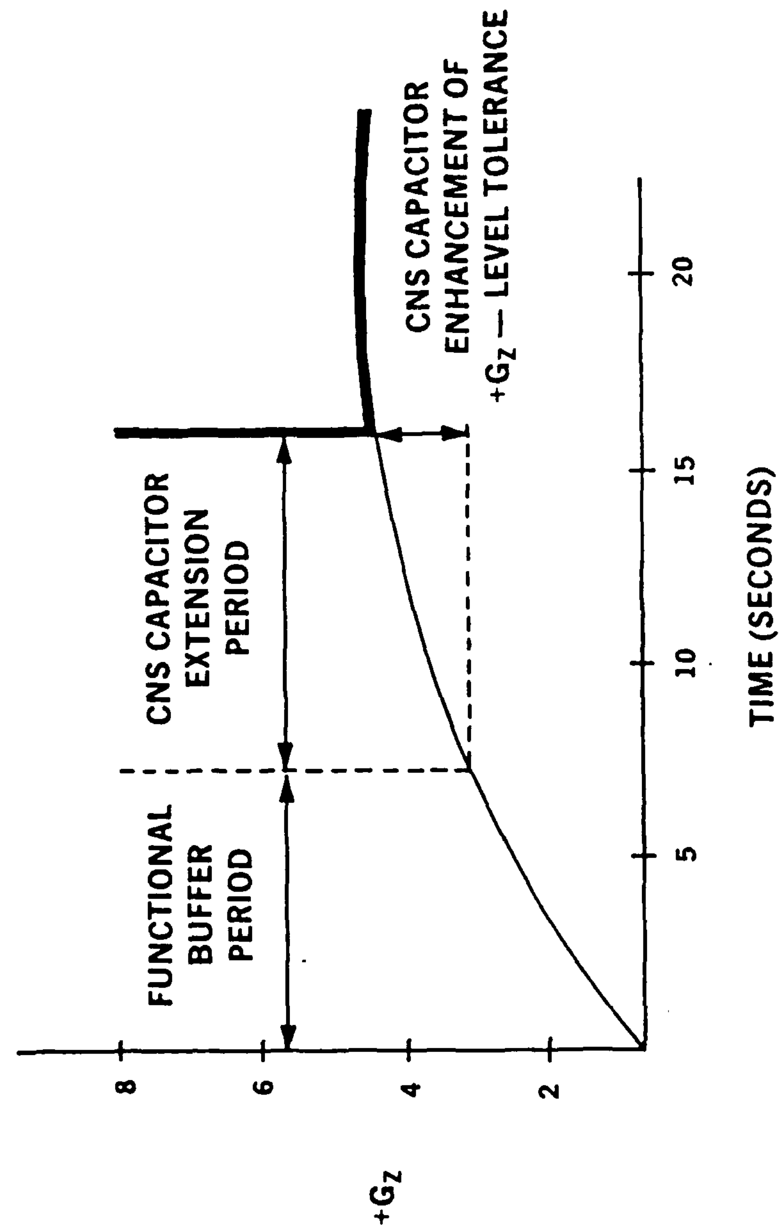

FIGURE 11. Dual Tolerance Enhancing Effects Resulting from Extension of the Functional Buffer Period.

mobilized. $+G_z$-level tolerance would then be increased (cardiovascular reflex compensation) by at least 1.5G, an amount equal to the protection provided by the current anti-G suit.

A CNS functional buffer period capacitor could be pursued along several lines. For instance, a biochemical CNS oxygen capacitor which would hold extra oxygen until the oxygen supply was inadequate to meet the demand at which time the additional oxygen would be released. Although the search for a mechanism, biochemical alteration, or pharmacologic agent to act as a CNS capacitor may seem overly ambitious, certainly the small amount of time, 5-7 seconds (or less), which would be extremely beneficial to fighter aircrew, makes this task seem more attainable. Longer buffer periods would be of even greater benefit and remain a research goal.

AN ADVANCED DEFINITION OF $+G_z$ TOLERANCE

Today nearly everyone, except the operational acceleration scientist, considers "$+G_z$-tolerance" to have a single meaning represented by a single value. This lack of understanding presents great difficulty for the acceleration scientist, especially when discussing $+G_z$ research with aircrew or other non-acceleration scientists. For good reason, the pilot is familiar with the effects of $+G_z$ when flying his aircraft. If asked, he would probably say $+G_z$ tolerance simply means "how many Gs I can pull without losing vision or going unconscious." For the acceleration scientist $+G_z$-tolerance is more complex, especially when evaluating the human response to $+G_z$-stress and when trying to develop advanced anti-G equipment and protective techniques. Although implicit in much of the research that is performed, an advanced definition scheme for $+G_z$-tolerance has not been established. To this end, the following discussion is provided to effect a unified understanding of what constitutes overall tolerance to $+G_z$-stress.

$+G_z$ tolerance can be divided into at least four different types of tolerance as shown in Figure 12. This $+G_z$-tolerance scheme encompasses the spectrum of important physiologic and aeromedical considerations that impact operational flying. As previously indicated, the pilot understands what he means when he wants to know how to enhance his $+G_z$-tolerance. The aerospace medicine physician on the other hand, must consider all the aspects of $+G_z$-tolerance that result in the pilot's ability to perform in the $+G_z$ aerial combat environment. It is of vital importance to recognize that operational $+G_z$-tolerance is complex and the appropriate type of $+G_z$- tolerance must be considered in each experimental investigation. All aspects must be investigated so that optimal protection can be afforded fighter aircrew in a combat environment. It is unfortunate that much acceleration research is performed without full investigation of the breadth of all types of $+G_z$-tolerance. The scope of such research becomes limited as related to its usefulness to combat aircrew.

$+G_z$-LEVEL TOLERANCE

This tolerance comes the closest to being what has been usually considered to represent traditional $+G_z$-tolerance. It is measured in $+G_z$- units. The variations of onset rate and duration at peak G make this type of tolerance dependent on each of these variables. In defining $+G_z$-level tolerance the onset rate and duration must therefore be given. For more gradual onset rates the $+G_z$-level tolerance is dependent on cardiovascular reflexes, whereas for more rapid onset rates it is strictly dependent on hydraulic considerations. These measurements have generally been made on relaxed subjects. $+G_z$-level tolerance measurements made with the subject performing some type of anti-G straining maneuver are also made but are dependent on the ability to perform an optimum maneuver, motivation, and to a certain extent on endurance.

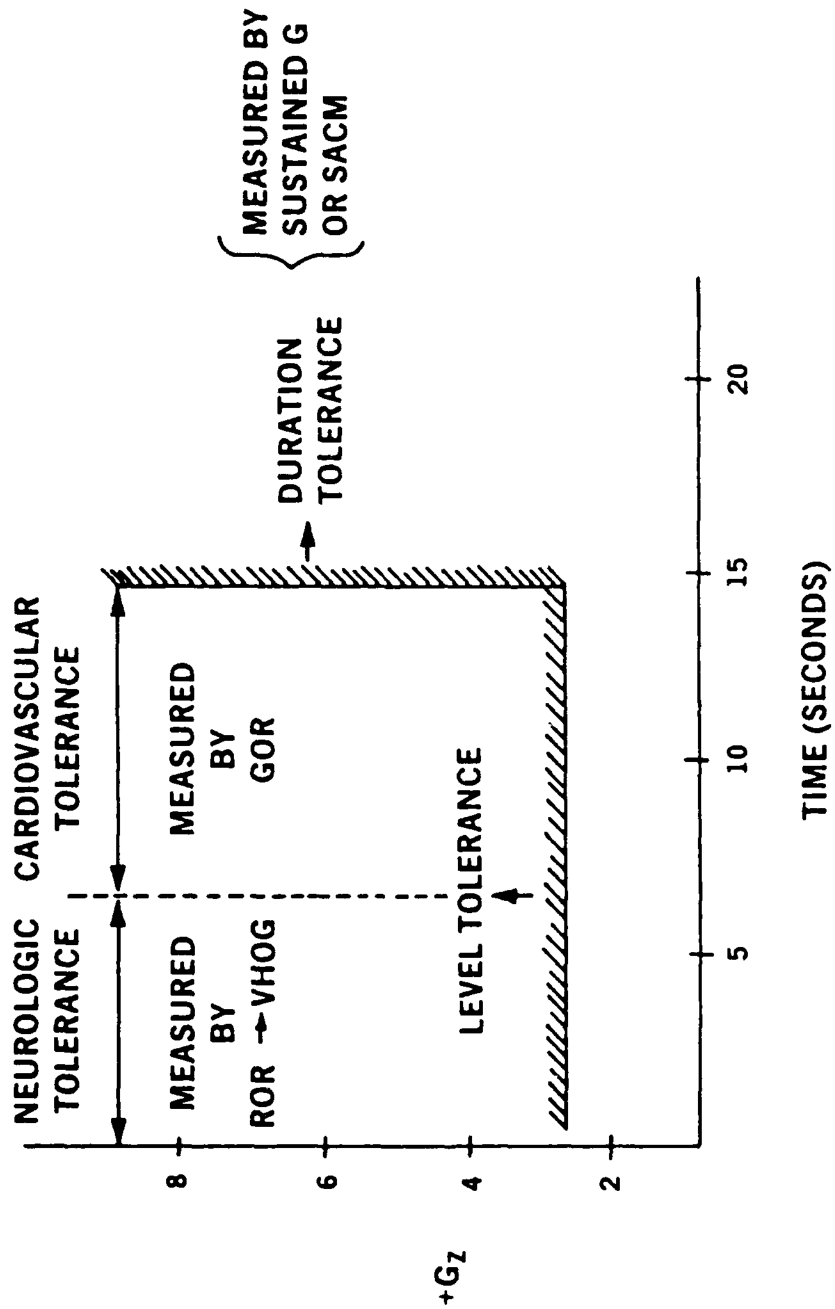

FIGURE 12. Measurement of the Four Types of $+G_Z$-Tolerance.

+G_z-DURATION TOLERANCE

Duration tolerance is important when considering the ability to tolerate acceleration over time and is based primarily on fatigue and/or discomfort as endpoints. Toleration at a particular level of +G_z for a certain length of time is measured. Simulated aerial combat maneuvering (SACM) profiles of variable +G_z levels for various periods of time are also used to determine this type of tolerance (1). The fatigue endpoints include not only fatigue, but, in addition, pain and the secondary effects of fatigue which result in visual symptoms and G-LOC. Although +G_z onset rates must be stated, this type of tolerance generally requires a description of the area under the +G_z level-time profile curve. Tolerance is given as a length of time or the integrated total area under the curve. Duration tolerance has been most useful in determining the effects of anti-G protective equipment and the benefits of physical conditioning.

+G_z-CARDIOVASCULAR TOLERANCE

This type of tolerance is dependent on the adequacy of the cardiovascular response to +G_z-stress. It overlaps somewhat with +G_z-level tolerance when using gradual onset +G_z stress. It also is dependent on a normal cardiac rate and rhythm response. +G_z-cardiovascular intolerance is manifest by such responses as dysrhythmias which compromise the ability to tolerate +G_z such as ventricular tachycardia or prolonged sinus pauses. As such this type of tolerance has been more of an aeromedical concern. +G_z-cardiovascular tolerance has been shown to be altered in some highly aerobically trained individuals. It is measured by a normal rate of change of heart for a given +G_z-onset rate and by the absence of cardiac rate and rhythm disturbances.

+G_z-NEUROLOGIC TOLERANCE

The +G_z-neurologic tolerance is generally determined using very high onset +G_z. It is determined by the length of time the CNS can function without adequate blood flow (i.e., the CNS functional buffer period). This type of tolerance is frequently what is relied upon during rapid or very high onset exposure to high +G_z for short duration. It is dependent on the length of time the CNS critical blood flow threshold is exceeded. It is measured in seconds.

OPERATIONAL +G_z-TOLERANCE FOR FIGHTER AIRCRAFT AIRCREW

Tolerance to +G_z in the operational environment is a complex combination of the various tolerances. Different phases of aerial combat maneuvering make certain specific types of tolerance more important at different times. The basic tolerance to very high onset, sustained high +G_z is initially dependent on the CNS functional buffer period and how rapidly the state of exceeding the threshold for inadequate blood flow to the CNS is reached. The onset rate which virtually instantaneously reduces CNS blood flow beyond the threshold for continuous function is defined as VHOG. At +G_z onset rates equal to or greater than this, blood flow to the CNS is reduced instantaneously and the tolerance becomes immediately determined by the CNS functional buffer period (if protective equipment and techniques fail to provide enough +G_z-level tolerance enhancement). Sustained +G_z must be maintained to exceed the functional buffer period, in the average individual this is approximately 7s. Although accurate definition of the exact onset rate where the instantaneous critical reduction of CNS blood occurs has yet to be accomplished, it can be estimated to begin somewhere around 2 G/s.

Initial sustained VHOG exposure becomes independent of the +G_z level once above an individuals hydraulic +G_z-level tolerance with protective equipment and techniques, unless the cardiovascular reflexes are able to respond rapidly enough to effect a rescue. Since cardiovascular reflexes evidently require 10-15s to adequately respond, and even then only confer an additional 1.5 G_z above the hydraulic +G_z-level tolerance; initial tolerance is independent of cardiovascular reflexes. Even if cardiovascular reflexes were able to respond before exceeding the functional buffer period, $+G_z$-level tolerance would only be increased to approximately $+5\ G_z$ unprotected. Above this level $+G_z$-level tolerance is determined exclusively by the functional buffer period. Between the hydraulic $+G_z$-level tolerance limit and the $+G_z$-cardiovascular tolerance limit the pilot is unlikely to be rescued due to the mismatch in time constants for the CNS functional buffer period (7s) and the cardiovascular response time (10-15s). Tolerance then is determined by the CNS functional buffer period with VHOG to levels above the hydraulic tolerance limit.

Above the hydraulic tolerance limit VHOG results in unconsciousness without warning since both brain and retinal perfusion are simultaneously halted and therefore unrecognizably fail simultaneously. Overall then, the initial operational tolerance of fighter aircrew becomes dependent on $+G_z$ level tolerance, $+G_z$-neurologic tolerance, to a certain extent the $+G_z$- cardiovascular tolerance and related protective equipment and anti-G straining maneuver effectiveness. After successfully achieving high $+G_z$, the aircrews ability to sustain $+G_z$ becomes dependent on $+G_z$-duration tolerance. The ability to perform the anti-G straining maneuver efficiently for a lengthy period being determined to a great extent by muscular endurance. $+G_z$-cardiovascular tolerance is critical throughout the exposure. A rate or rhythm disturbance that compromises cardiac output reduces tolerance at any point during or after the exposure. Exaggerated slowing of the rhythm post-G stress is potentially compromising by virtue of the possible need to repeatedly go to high $+G_z$ as in aerial combat maneuvering. Operational $+G_z$-tolerance is therefore complex and requires adequate tolerance to the combination of all four types of tolerance. Individual aircrew tolerance to the acceleration environment of aerial combat and evaluation of $+G_z$ protective equipment and techniques must be performed considering all the type of $+G_z$-tolerance that may ultimately affect performance.

The above description of the concept of overall $+G_z$ tolerance may not be all inclusive but should, at least, represent a beginning effort for the full definition of the important aspects of what constitutes operation tolerance and the required measurements for acceleration research.

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